Pallepati Srujana et al, 2026, Vol.14, No.4: 73-78

International Journal of Chemistry and Pharmaceutical Sciences

https://doi.org/10.30904/j.ijcps.2026.5011 | E-ISSN: 2321-3132

Evaluation of Neuroprotective Activity of Selected Plant Extract in Mouse Model

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Received: 30-01-2026 | Revised: 21-02-2026 | Accepted: 28-03 2026 | Published: 30-04-2026

Abstract:

Neurodegenerative diseases such as Alzheimer's disease (AD), Parkinson's disease (PD), and other related disorders represent a major global health challenge due to their progressive, chronic, and incurable nature. Their pathogenesis involves multifactorial mechanisms, including oxidative stress, cholinergic dysfunction, mitochondrial impairment, protein aggregation, and neuroinflammation. Conventional therapies, such as cholinesterase inhibitors and NMDA antagonists, provide only symptomatic relief and are associated with safety concerns and limited efficacy. This scenario underscores the need for novel neuroprotective agents that are safe, effective, and multi-targeted. In this context, the present study investigated the neuroprotective potential of fresh fruit juice of *Actinidia deliciosa* (FJAD) against $AlCl_3$-induced neurotoxicity in Sprague Dawley rats. The findings strongly suggest that FJAD offers significant neuroprotection through antioxidant, anti-cholinesterase, and neuro-restorative pathways, comparable to the standard drug Vitamin E.

Keywords: *Neuroprotective Activity, Plant Extract, NMDA, FJAD, Alzheimer's disease*

Introduction

Plant Profile

Botanical Name: *Actinidia deliciosa* (fuzzy kiwifruit)

Family: Actinidiaceae

Origin: Native to southern China; widely cultivated globally.

Habitat: Found at altitudes between 600–2000 m in China, Japan, and Russian Far East.

Scientific Classification

- **Kingdom:** Plantae
- **Division:** Magnoliophyta
- **Class:** Magnoliopsida
- **Subclass:** Magnolidae
- **Order:** Ericales
- **Superorder:** Asteranae
- **Family:** Actinidiaceae
- **Genus:** Actinidia
- **Species:** *A. deliciosa*

Fresh fruits of *Actinidia deliciosa* were washed thoroughly to remove adhering impurities, ground into paste using a homogenizer, and squeezed through muslin cloth. The obtained juice was centrifuged at 5000 rpm for 15 min at room temperature to yield a clear extract.

% Yield of Juice was calculated as:

% Yield = Quantity of juice obtained (mL)/ {Weight of fruit slices taken (g) X 100

Determination of Weight/Volume

- **Procedure:**
  1 mL of juice was transferred into a pre-weighed specific gravity bottle (W_0). Its weight with juice was recorded as $\mathbf{W_1}$.
- The bottle was oven-dried (40–45°C) until semi-solid consistency, then weighed as $\mathbf{W_2}$.

The difference in weights i.e,W1-W2 gives the weight/volume of the juice.

This value was used to prepare appropriate concentrations for in vivo studies.

Fig.1: Preparation of Fruit Juice

Dose Preparation

Based on wt/vol values, fresh juice doses of 200 mg/kg and 400 mg/kg body weight were prepared for administration.

Experimental Animals

- **Species:** Sprague Dawley rats (male)
- **Age:** 12–15 months
- **Weight:** 150–200 g
- **Source:** VAB Biosciences, Hyderabad
- **Housing:** Polypropylene cages, 27 ± 2°C, RH 44–56%, 12-h light/dark cycle, free access to chow and water.
- **Ethics:** Approved by IAEC, Teena Biolabs Pvt. Ltd., Hyderabad, under CPCSEA guidelines.

Materials and Methods

Table 1: Instruments/Apparatus used

S.No.	Instrument/Apparatus	Source
1	Test tubes	Borosil
2	Centrifuge tubes	Tarsons
3	Cooling centrifuge	REMI
4	Digital balance	ELICO
5	UV-Vis Spectrophotometer	Shimadzu
6	pH Meter	Elico LI 127
7	Mortar and pestle	Swasthic Scientific Co., Mumbai

Table 2: Treatment Groups

Group	Treatment	Purpose
Normal	Standard chow + water	Baseline physiology
Control	$AlCl_3$ (150 mg/kg p.o.)	Neurotoxicity induction
Standard	Vitamin E (50 mg/kg from day 20)	Positive control (antioxidant)
Test I	FJAD (200 mg/kg from day 20) + $AlCl_3$	Evaluate therapeutic effect
Test II	FJAD (400 mg/kg from day 20) + $AlCl_3$	Dose-dependent effect

Experimental Design

- Blood collection: Retro-orbital route under light ether anesthesia.
- Brain collection: Post-euthanasia; one hemisphere used for biochemical assays, other fixed in formalin for histology.

Behavioral Assessments

1. **Locomotor activity:** Actophotometer, 10 min/session.
2. **Muscle strength:** Rota-rod test at 20–25 rpm, cut-off 5 min.
3. **Elevated Plus Maze:** Spatial memory (Initial Transfer Latency vs. Retention Transfer Latency).
4. **Morris Water Maze:** Spatial learning, probe trials, and visible platform tests with video tracking.

Biochemical Estimations

- **Acetyl cholinesterase (AChE) activity** — Ellman's method (412 nm, UV-Vis spectrophotometer).
- **Lipid Peroxidation (MDA levels)** — Thiobarbituric Acid Reactive Substances (TBARS) method.
- **Antioxidant defense system:** SOD, CAT, GPx, and reduced GSH measured in homogenates.

Histological Assessment

After completion of behavioural and biochemical studies, the brains of animals from all experimental groups were subjected to histopathological evaluation in order to assess neuronal integrity and hippocampal lesions.

Tissue Fixation: The excised brains were perfusion-fixed with 4% paraformaldehyde in 0.1 M phosphate buffer (pH 7.4). Tissues were post-fixed overnight at 4°C in the same fixative. For embedding, tissues were dehydrated in graded alcohols, cleared in xylene, and embedded in paraffin blocks.

Fixation was essential because it:

a) Hardened the tissues by coagulating cellular proteins,
b) Prevented autolysis and putrefaction,
c) Preserved cellular and tissue architecture, and
d) Prevented shrinkage and distortion.

Commonly, **10% formalin** was used as a routine fixative.

Sectioning and Staining

Paraffin blocks were sectioned at 5–6 µm thickness using a rotary microtome. Sections were mounted on clean slides and stained with hematoxylin and eosin (H&E) for general histoarchitecture.

H&E staining procedure:

1. Deparaffinization in xylene for 5–10 min.
2. Rehydration through graded alcohol to water.
3. Staining with hematoxylin for 3–4 min.
4. Washing in tap water and differentiation if required.
5. Counterstaining with 0.5% eosin for 15–30 seconds until a light pink background was observed.
6. Dehydration in graded alcohol, clearing in xylene, and mounting with DPX or Canada balsam.
7. Slides were observed under 10× and 40× magnification for neuronal morphology and hippocampal lesions.

Evaluation of Pro-Oxidant Status (Lipid Peroxidation Assay)

To quantify oxidative stress in brain tissues, malondialdehyde (MDA) levels, a stable end-product of lipid peroxidation, were measured following the Slater and Sawyer method.

Principle

MDA reacts with thiobarbituric acid (TBA**)** under acidic and high-temperature conditions to form a pink chromogen, which is measured spectrophotometrically at 535 nm. The absorbance is directly proportional to the extent of lipid peroxidation.

Reagents

1. **Thiobarbituric acid (0.67%)** in 1 M Tris-HCl buffer, pH 7.0.
2. **Trichloroacetic acid (20%)** prepared in distilled water.
3. **Standard MDA solution (0.25 nmol):** Prepared from 1,1,3,3-tetraethoxypropane in Tris-HCl buffer (pH 7.0). Used for calibration curve preparation.

Procedure

1. 2 ml of brain tissue homogenate was mixed with 2 ml of 20% trichloroacetic acid and kept on ice for 15 min.
2. The mixture was centrifuged at 3000 rpm for 10 min.
3. 2 ml of clear supernatant was combined with 2 ml of 0.67% thiobarbituric acid.
4. Samples were heated in a boiling water bath for 10 min, then cooled rapidly on ice.
5. Absorbance was measured at **535 nm** using a UV-visible spectrophotometer.
6. MDA concentrations were calculated using the standard calibration curve and expressed as nmol MDA/mg protein.

4.12 Statistical Analysis

All experimental data were expressed as Mean ± Standard Error of Mean (SEM) for each group (n = 6 animals per group). The data were subjected to one-way analysis of variance (ANOVA) to evaluate the significance of differences among the various treatment groups. Where the ANOVA indicated statistical significance, Tukey's multiple comparison post-hoc test was applied to compare individual groups.

The level of statistical significance was set at:

- $p < 0.05$ – significant
- $p < 0.01$ – very significant
- $p < 0.001$ – highly significant

Graphical representation of data (bar graphs, line plots) was carried out using GraphPad Prism software (version 8.2.3,

GraphPad Software Inc., San Diego, USA). Results were interpreted based on the degree of significance compared to the $AlCl_3$ control group.

Result and Discussion

Blood Parameters

The effect of $AlCl_3$ and treatment with fresh juice of *Actinidia deliciosa* (FJAD) and Vitamin E on hematological parameters is presented in **Table 5.1**.

- **$AlCl_3$ exposure** caused a marked reduction in total WBC count (2.55 $\times 10^3/\mu l$) and platelets (650 $\times 10^3/\mu l$) compared to normal values (5.51 $\times 10^3/\mu l$ and 710 $\times 10^3/\mu l$, respectively).
- Reticulocyte count was elevated in the control group, indicating hematopoietic stress.
- **FJAD treatment** (200 and 400 mg/kg) significantly restored WBC, platelet count, and reticulocyte levels toward normal.

Vitamin E also normalized blood indices but FJAD (especially 400 mg/kg) produced better recovery of WBC levels.

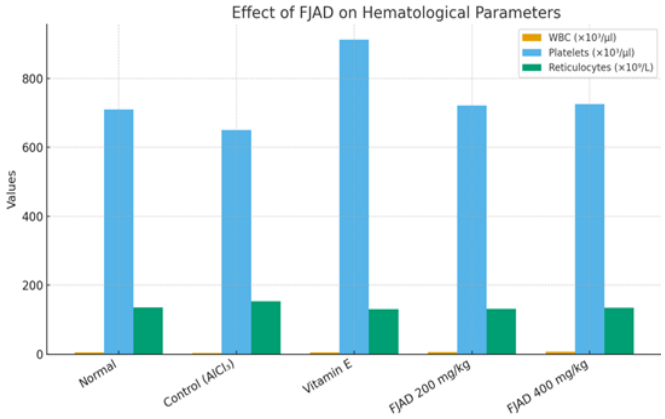

Fig.2: Bar graph of hematological data

Behavioral Studies

$AlCl_3$ significantly impaired locomotor activity, motor coordination, and learning/memory functions compared to normal animals. Treatment with FJAD (200 and 400 mg/kg) significantly improved these parameters in a dose-dependent manner**.**

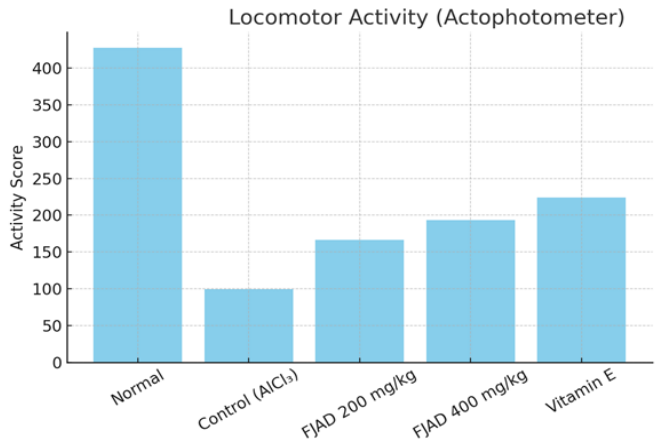

Fig.3

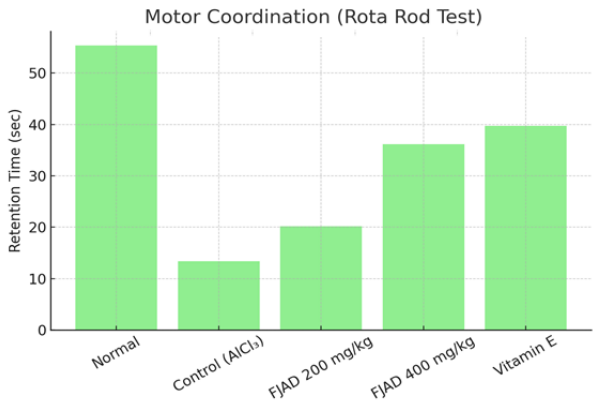

Fig.4

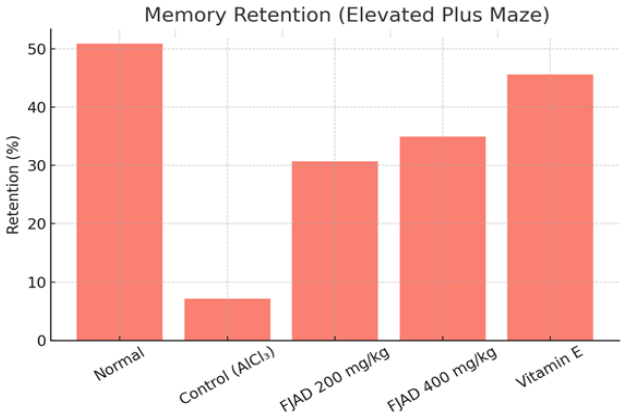

Fig.5

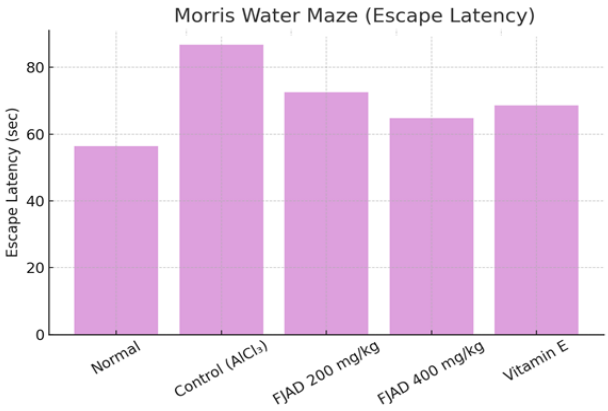

Fig.6

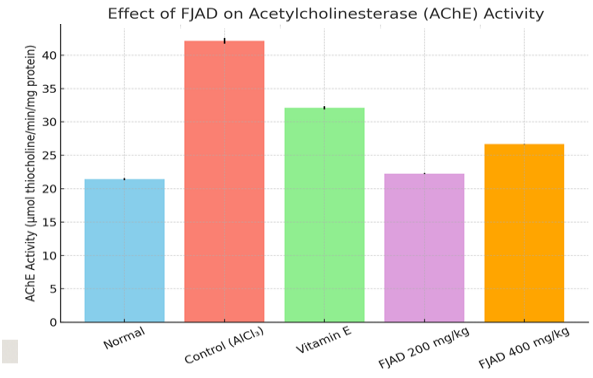

Fig.7

Discussion

Neurodegenerative diseases such as Alzheimer's disease (AD), Parkinson's disease (PD), and other related disorders represent a major global health challenge due to their progressive, chronic, and incurable nature. Their pathogenesis involves multifactorial mechanisms, including oxidative stress, cholinergic dysfunction, mitochondrial impairment, protein aggregation, and neuroinflammation. Conventional therapies, such as cholinesterase inhibitors and NMDA antagonists, provide only symptomatic relief and are associated with safety concerns and limited efficacy. This scenario underscores the need for novel neuroprotective agents that are safe, effective, and multi-targeted. In this context, the present study investigated the neuroprotective potential of fresh fruit juice of *Actinidia deliciosa* (FJAD) against $AlCl_3$-induced neurotoxicity in Sprague Dawley rats. The findings strongly suggest that FJAD offers significant neuroprotection through antioxidant, anti-cholinesterase, and neuro-restorative pathways, comparable to the standard drug Vitamin E.

Hematological Findings

$AlCl_3$ administration led to notable hematological alterations, particularly suppression of WBC count and platelet reduction, along with elevated reticulocyte counts indicative of hematopoietic stress. These findings are consistent with previous reports where aluminum exposure caused bone marrow suppression, oxidative stress, and altered immune responses.

FJAD significantly restored WBC, platelet counts, and reticulocyte values toward normal. This suggests that the juice exerts immunoprotective and hematopoietic-stabilizing effects, potentially mediated by its rich antioxidant profile (ascorbic acid, polyphenols, flavonoids). Unlike synthetic drugs, which primarily target specific pathways, FJAD maintained a broader hematological balance without altering red cell indices (MCV, MCH, MCHC).

Behavioral Outcomes: Neurobehavioral tests are critical for validating neuroprotective effects in experimental models:

- Locomotor activity was severely impaired by $AlCl_3$, reflecting generalized neurotoxicity. FJAD restored activity in a dose-dependent manner, similar to Vitamin E, indicating protection of motor pathways.
- Motor coordination (Rota rod test): $AlCl_3$ drastically reduced muscle coordination and balance. FJAD at 400 mg/kg significantly improved performance, suggesting protection of neuromuscular and cerebellar circuits.
- Elevated Plus Maze: $AlCl_3$ induced marked memory deficits, consistent with cholinergic dysfunction. FJAD significantly improved retention, indicating enhanced learning and memory consolidation.
- Morris Water Maze: Escape latency was prolonged by $AlCl_3$, confirming spatial learning impairment. FJAD shortened escape times, particularly at 400 mg/kg, suggesting restoration of hippocampal-dependent memory.These behavioral improvements align with the traditional use of kiwifruit in cognitive and adaptogenic health and highlight its multi-target efficacy.

Acetylcholinesterase Activity

Cholinergic dysfunction is central to AD pathology. In the present study, $AlCl_3$ significantly elevated AChE activity, reducing acetylcholine levels and impairing neurotransmission. Treatment with FJAD, especially at 200 mg/kg, almost completely normalized AChE activity. This effect may be attributed to bioactive compounds such as flavonoids, phenolics, and quinic acid, which have been shown to inhibit AChE activity and enhance cholinergic function.The findings are in agreement with prior studies where plant polyphenols improved memory by inhibiting AChE and preserving synaptic function. Thus, the anti-AChE activity of FJAD provides a strong mechanistic basis for its cognitive-enhancing properties.

Antioxidant Effects: Lipid Peroxidation

Oxidative stress is a primary contributor to neuronal injury in AD and other neurodegenerative conditions. In this study, $AlCl_3$ significantly elevated MDA levels, indicating lipid peroxidation and neuronal membrane damage. FJAD treatment reduced MDA levels in a dose-dependent manner, with 400 mg/kg showing strong antioxidant protection comparable to Vitamin E. This is consistent with earlier reports of kiwifruit's high antioxidant capacity, attributed to its ascorbic acid, carotenoids, and phenolic compounds. By reducing oxidative stress, FJAD likely prevents downstream mitochondrial dysfunction and apoptotic signaling, thereby protecting neuronal integrity.

Histopathological Findings

Histological analysis confirmed the biochemical and behavioral results. $AlCl_3$-treated rats exhibited neuronal degeneration, mild inflammatory infiltration, and vascular changes. FJAD-treated groups, particularly at higher dose, showed preserved pyramidal and neuroglial cells with minimal inflammatory changes. These protective effects paralleled Vitamin E, indicating that FJAD preserves brain architecture and prevents neurodegenerative changes.

6.6 Mechanistic Insights

The observed neuroprotective effects of FJAD may be explained by the following mechanisms:

1. Antioxidant activity: Scavenging free radicals and reducing lipid peroxidation.
2. Cholinergic modulation: Inhibition of AChE, preserving acetylcholine levels and improving synaptic function.
3. Immunoprotection: Restoration of WBC and platelet counts, preventing systemic toxicity of $AlCl_3$.
4. Neuroprotection: Structural preservation of hippocampal neurons, consistent with improved memory and motor functions.

Thus, FJAD exerts multi-targeted neuroprotection, making it a promising candidate for further investigation in chronic neurodegenerative disease models.

Comparison with Standard (Vitamin E)

Vitamin E, a well-established antioxidant, was used as a standard reference. While Vitamin E showed strong antioxidant and neuroprotective effects, FJAD at 400 mg/kg produced comparable results across behavioral, biochemical, and histological parameters. Interestingly, the 200 mg/kg dose was particularly effective in AChE inhibition, highlighting possible dose-dependent differential mechanisms. This suggests that FJAD may not only act as an antioxidant like Vitamin E but also exert additional cholinergic and immunomodulatory effects, offering a broader neuroprotective profile.

Limitations and Future Perspectives

Although the present study provides strong evidence of neuroprotection, some limitations remain:

- The study focused on acute $AlCl_3$-induced neurotoxicity; chronic models of AD should be evaluated.
- Detailed phytochemical profiling of FJAD is required to identify active compounds.
- Pharmacokinetic studies are essential to confirm brain penetration of bioactives.
- Long-term safety and dose optimization need further exploration.

Future studies should also explore molecular markers such as BDNF, NF-κB, Nrf2, and apoptotic regulators (Bcl-2/Bax ratio, caspase-3) to provide deeper mechanistic insights. The present study demonstrates that fresh fruit juice of *Actinidia deliciosa* protects against $AlCl_3$-induced neurotoxicity in rats by improving behavioral performance, restoring cholinergic function, reducing oxidative stress, and preserving brain histology. Its effects were dose-dependent and comparable to Vitamin E, confirming its potential as a safe and natural neuroprotective agent. This investigation provides a scientific basis for the ethnomedicinal use of kiwifruit and underscores its promise as a candidate for complementary and alternative therapy in neurodegenerative disorders.

Conclusion

The present investigation was undertaken to evaluate the neuroprotective potential of fresh fruit juice of *Actinidia deliciosa* (FJAD) against $AlCl_3$-induced neurotoxicity in Sprague Dawley rats. Based on the results obtained, the following conclusions can be drawn:

1. **Hematological protection:**
   $AlCl_3$ induced significant alterations in hematological parameters, particularly a reduction in WBC counts and platelet levels, along with stress-related reticulocytosis. Treatment with FJAD restored these parameters toward normal, indicating immunoprotective, hematopoietic-stabilizing effects.
2. **Behavioral improvements:**
   $AlCl_3$ markedly impaired locomotor activity, motor coordination, and cognitive functions as evidenced by actophotometer, rota rod, elevated plus maze, and Morris water maze tests. FJAD significantly improved these behavioral outcomes in a dose-dependent manner, with the higher dose (400 mg/kg) performing comparably to Vitamin E.
3. **Cholinergic modulation:** $AlCl_3$ exposure resulted in elevated acetylcholinesterase activity, indicating cholinergic dysfunction. FJAD significantly inhibited AChE activity, particularly at 200 mg/kg, thereby preserving acetylcholine levels and improving memory function.
4. **Antioxidant activity**: Brain lipid peroxidation (MDA levels) was significantly elevated in $AlCl_3$-treated rats, confirming oxidative stress. FJAD markedly reduced MDA concentrations in a dose-dependent fashion, with the 400 mg/kg dose showing strong antioxidant activity similar to Vitamin E.
5. **Histopathological protection:**
   Microscopic analysis of brain tissues showed neuronal degeneration and inflammatory changes in $AlCl_3$-treated rats. FJAD treatment preserved neuronal structure and reduced inflammatory infiltration, further confirming its neuroprotective action.

Conflict of Interests

The authors declare no conflict of interest

Ethics Approval: Not applicable

Funding: This study received no specific funding from public, commercial, or not for profit funding agencies.

AI Tool Declaration

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

Data Availability

Data will be available on request

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